REMARKS/ARGUMENTS

Favorable reconsideration is respectfully solicited in light of the foregoing amendments and the remarks which follow.

In response to the indefiniteness rejection under 35 U.S.C. 112, second paragraph, claim 1 has been amended such that the poly(ethylene oxide) chain is now defined "as consisting" of 30 to 100 ethylene oxide units, so that the scope of the compound is now clearly defined. A similar amendment has been introduced in claim 3.

With regard to the enablement rejection under 35 U.S.C. 112, first paragraph, the Examiner asserts that with regard to Y and Y', terms such as, for example, ester, encompass a moiety of any structural formula containing an ester as part of its structure. To overcome this objection, in claim 1 the expressions "ester, amide...amine function" have been replaced by their chemical formula, so that the scope of the claims is now limited to these structures. Accordingly, these claims are fully enabled.

It appears that claim 16 has not been taken into account by the Examiner. It is requested that the finality of the rejection be withdrawn so that claim 16 can be considered on the merits.

With regard to claims 9 to 11 and 13, the Applicant disagrees with the Examiner's point of view and requests reconsideration by the Examiner. Please refer to the prior demonstration provided in the amendment dated September 16, 2008, p. 13, paragraph 4 - p. 14, paragraph 3. The disclosure shows that some compounds illustrated in the examples trap free radical activity, and have neuroprotective activity on nerve-muscle cocultures. And pathological conditions linked to oxidative stress and the formation of oxygen-containing free radical species have been listed by Cross C.E., *Arch, Intern, Med.* (1987) **107,** 526-545 and by Anderson K.M., Ells G., Bonomi P., Harris J.E., *Medical Hypotheses* (1999) **52,** 53-57. Among these have been listed: immune and inflammatory diseases, the ischemiareperfusion syndrome, atherosclerosis, Alzheimer's and Parkinson's diseases, lesions due to UV and ionizing radiations, certain forms of chemical carcinogenesis and cellular aging.

The therapeutic effect of nitrones in the reduction and prevention of the damage caused by free radicals in biological systems was demonstrated in 1990 by Oliver C., Starte-Read

P., Stadman E., Liu G., Carney .1., Ploys R. Proc. Acad. USA (1990) 87, 5144-5147. These authors demonstrated a decrease in the damage caused by cerebral ischemia in gerbils after a-C-phenyl-N-tort-butylnitrone (PBN) had been injected. Cerebral isehemias are accompanied by a large increase in the production of free radicals, which were trapped by the PBN, thereby forming spin adducts which were much more stable and therefore less reactive and toxic. PBN is the spin trap to which the largest number of biological studies have related. And the results obtained in biological tests for the compounds of the invention are superior to those obtained by PNB (see p. 17-p. 20).

Enablement was based on literature disclosure of analogous compounds having proven efficient *in vivo,* and on test methods disclosed therein which gave positive results with prior art nitrones.

As additional evidence, please find submitted herewith and listed on the accompanying form 1449/PTO several additional scientific publications regarding the biological efficiency of the molecules of the invention. The tests disclosed therein have been performed *in vivo* and are positive.

Citation (1): S. Tanguy, G. Durand, C. Reboul, A. Polidori, B. Pucci, M. Dauzat, P. Obert, (2006), Protection against reactive oxygen species injuries in rat isolated perfused hearts: effect of LPBNAH, a new amphiphilic spin-trap derived from PBN". *Cardiovascular Drugs and Therapy*, Vol. 20, pages 146-149. This document discloses results regarding LPBNAH which is named nitrone Al in the present application. Protective effect *in viva* against ROS injuries was noted for these compounds.

Citation (2): Burkhard Poeggeler, Gregory Durand, Ange Polidori, Miguel Pappolla, Ignacio Vega-Naredo, Ana Coto-Montes, Jutta Böker, Rudiger Hardeland and Bernard Pucci. (2005) *Journal of Neurochemistry*, 2005, 95, 962-973 Mitochondrial: Neuroprotection and life extension by the new amphiphilic nitrone LPBNAH acting as a highly potent advanced antioxidant agent: This document discloses in vivo testing of nitrone Al (LPBNAH see figure 1 of the publication).

Those tests demonstrate that this compound is unique in its exceptional anti-ageing efficacy, being one order of magnitude more potent than any other compound previously tested on rotifiers. The nitrone protected these aquatic animals against the lethal toxicity of hydrogen peroxide and doxorubicin and greatly enhanced their survival when co-administrated with the oxidotoxins. These findings indicate that amphiphilic antioxidants have a great potential as neuroprotective agents in preventing the death of cells and organisms exposed to enhanced oxidative stress and damage.

Citation (3): Taketoshi Asanuma, Hironobu Yasui, Osamu Inanami, Kenji Waki, Momoko Takahashi, Daisuke Iizuka, Taketo Uemura, Gregory Durand, Ange Polidori, Yasuhiro Kon, Bernard Pucci, Mikinori Kuwabara. *Chemistry & Biodiversity*. (2007), 4, 2253-2267. This document discloses tests performed *in vivo* on rats with nitrone A2 (LPBNSH figure 1 of the publication). These tests are based on spin-trap properties of nitrone A2 and prove to have liver-protective effect against fulminant hepatitis with jaundice. Therefore these compounds are clinically promising.

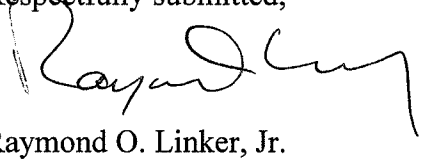
It should be recalled that *in vivo* testing cannot be performed before obtaining *in vitro* results for reasons of costs notably. And on the other hand it is not possible to delay unreasonably the filing of an application once one has obtained *in vitro* results which show very high probability of a confirmation *in vivo.*

This confirmation being provided demonstrates the assumptions on which the application was based were not unreasonable.

For these reasons, we submit that claims 9 to 11 and 13 are fully enabled. Reconsideration by the Examiner and withdrawal of the rejection under 35 U.S.C. 112, first paragraph, is solicited.

It is not believed that extensions of time or fees for net addition of claims are required, beyond those that may otherwise be provided for in documents accompanying this paper. However, in the event that additional extensions of time are necessary to allow consideration of this paper, such extensions are hereby petitioned under 37 CFR § 1.136(a), and any fee required therefore (including fees for net addition of claims) is hereby authorized to be charged to Deposit Account No. 16-0605.

Respectfully submitted,



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LEGAL02/31186333v1

ELECTRONICALLY FILED USING THE EFS-WEB ELECTRONIC FILING SYSTEM OF THE UNITED STATES PATENT & TRADEMARK OFFICE ON March 10, 2009.